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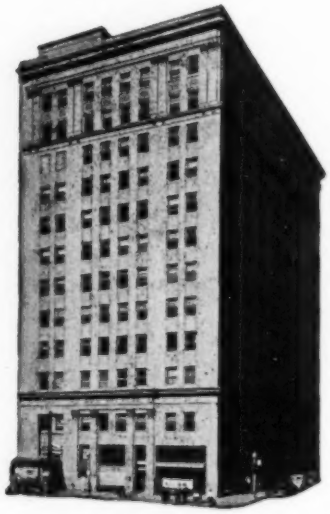

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E. GOLDFAIN, M.D.
Director

The JOURNAL Of The
OKLAHOMA STATE MEDICAL ASSOCIATION

VOLUME XLVI OKLAHOMA CITY, OKLAHOMA, FEBRUARY, 1953 NUMBER 2

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THE JOURNAL
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EDITORIALS

HOSPITAL AND MEDICAL SCHOOL COSTS

Ever mounting agitation about the cost of medical education and hospitalization has led to the question of Federal subsidy and government control. That this is not the answer is suggested by the long existing relatively high cost of maintaining our own federal hospitals.

Perhaps of greater significance is the experience of Britain's Ministry of Health under the existing National Health Service Act of 1948. Since the act went into effect, there has been more publicity about the overstaffing of hospitals and the rising costs.

The November 22 issue of the *British Medical Journal*[1] carries two significant editorials bearing on the costs of hospitals and medical education: 1. Reduction of hospital staffs. 2. Social medicine at Oxford. Under the first we learn with no surprise that the ". . . hard pressed officials of the Ministry are having to carry out the Treasury instructions," in spite of the fact the hospital enployees should ". . . feel that they are copartners in a joint enterprise and not just casual labor to be hired and fired according to the latest rule of the Treasury." Should not medicine and government in the United States be forewarned by Britain's present predicament.

How familiar the first sentence in the following quotation sounds to those who have been watching the growing centralization of power in our own government. And how important it is for us to contemplate the continuing tone of this editorial as it closes with the sad note of the Ministry's promises:

"What is to happen to those whose jobs will be thus brought to an end? The boards are obliged, Sir Russell points out, to find them alternative work, but even then, can anyone, least of all those affected, feel happy about the situation which has been allowed to develop? Will the boards be able to find alternative work in the face of an all round reduction of staffs? It is the answer to these questions which many will want to know, questions which consultation with the profession might have helped to answer in advance. The young and coming generation of consultants must reflect somewhat cynically on the exaggerated promises made by politicians in particular before the Health Service came into operation. There was then foretold in glowing terms the widely opening opportunities for consultants and specialists in the National Health Service. An increase by as much as 50 per cent was foreseen by some well-known apologists. The present incident to which Sir Russell draws attention is yet another illustration of the dangers of centralization. Those who are pressing for decentralization in the administration of the hospital services will be encouraged by this to redouble their efforts."

The editorial indirectly referring to medical education has to do with the closure of the Institute of Social Medicine at Oxford University. Economy demanded a review of its relative importance and its fate is well told in this paragraph:

"When the Institute of Social Medicine was founded at Oxford in 1942 the Nuffield Provincial Hospitals Trust made a grant of 100,000 pounds to cover the expenses of the Institute for 10 years. The grant has now come to an end, and the Board of the Faculty of Medicine at Oxford has recommended that the study be continued but at a lower level of expenditure, with a reader but without an institute. Evidently the board has had the difficult task of having to decide whether to abandon the institute or to cut down on other more vital projects. In a letter in which he discussed this dilemma Professor A. D. Gardner pointed out that, if Oxford were to ask the University Grants Committee to earmark a grant specially for social medicine, this 'would falsely imply that social medicine is of such unique importance to us that we must secure it whatever happens to other subjects.' Hard economic facts cannot be

ignored and doubtless the decision of the Board of the Faculty of Medicine was inevitable, but all who have an interest in Medicine in its widest aspects will deplore the passing of the Oxford institute."

1. *British Medical Journal*. No. 4794. Saturday, November 22, 1952. Page 1140.

SOCIALISM AND COMMUNISM

Apparently the leveling process is sweeping the world. Wherever it prevails, intelligence is imprisoned, incentive is throttled and personal initiative is usurped by the state.

What a dreary world with neither rich nor poor, good nor bad, wise nor foolish. What a grim level on which to live. What an asphyxiating atmosphere with no hope of resuscitation. Even hell has its gradations. Such an existence would be worse than Dante's lower circle.

Though we no longer have to carry our guns to the field to save our guts while we make corn for our grits, we are threatened with a fate that would be much worse than being shot on the spot.

Physicians should be the first to recognize the danger, set their stakes and lift their sights. Through education and public relations they should lead the fight against the annulling influence of this vicious political philosophy that is killing the world.

KNOWLEDGE, REASON AND JUSTICE

An informed judge, United States Circuit Court of Appeals, reasoning logically, puts the Hoxsey Cancer Clinic of Dallas out of business.

In medicine and in law, "a little knowledge is a dangerous thing." The Texas trial judge deficient in medical knowledge said, "Some it cures and some it does not cure, and some it relieves somewhat." The Judge of Appeals Court, after discussing tissue diagnosis of cancer, had the wisdom to say, ". . . the opinion of a layman as to whether he has cancer or whether he has been cured . . . is entitled to little weight.".

In the *Journal* of April, 1947, there is a long discussion of the Hoxsey hoax.

How long it takes for knowledge to sink in! How long for justice to arise! Thanks to a wise judge. Congratulations to all frustrated cancer victims. Goodbye, Mr. Hoxsey.

A TIP FOR THOSE IN CHARGE OF ADMISSIONS TO MEDICAL SCHOOLS

In Louisville, Kentucky, 1852, the first President of the Kentucky State Medical Association, William Loftus Sutton, giving advice to prospective preceptors, said:

"The private physician should absolutely refuse to receive into his office for instruction, any young gentleman whose natural intellect, moral character, and previous academic training, do not afford grounds to believe that he will make a respectable member of the profession."

THE CONSUMMATE DANGERS OF EDITORIAL COUNCIL

In the famous tragedy, "Antigone," Sophocles wrote, ". . . of all curses which cleave to man, ill council is the sovereign curse." Fortunately, the readers of the *Journal* are of such intellectual caliber they need not be informed of the heavy responsibility resting upon the shoulders of the *Journal's* Editorial Board. The Board must determine policies and accept responsibility for what is printed in the editorial columns. But, fortunately, for the members of the Editorial Board, the readers because of education and experience so nearly approach "the mature mind" they may be further informed but not too readily influenced by editorial pronouncements. Yet in view of the November national landslide including the surprising Oklahoma avalanche the Journal and its Editorial Board and the staff at the Executive Office of the State Association may with a certain degree of pride look forward to the death of the New-Fair-Deal when Eisenhower takes over January 2.

To some extent the belated political revolution may be considered as a justification of our long continued editorial policies against entrenched, power mad, bureaucrats in Washington and a vindication of our avowed cause, the freedom of both patient and physcician in their professional relationships, considering the socialistic psychology of the masses, the battle must continue.

So with the curse of ill council ever before us the Editorial Board carries on. Though reader council is subject to the same curse, please remember we crave constructive criticism even though it cleaves to the core.

Scientific Articles

BUTAZAOLIDIN IN RHEUMATIC DISEASES

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OKLAHOMA CITY, OKLA.

At the June, 1952, American Rheumatism Association convention preliminary reports on Butazolidin therapy were given by several workers. They observed favorable effects in rheumatic conditions such as gouty arthritis, Marie-Strumpel spondylitis, psoriatic arthritis and to some degree in osteoarthritis.

Some rheumatoid arthritis cases obtained good relief. Others did not react favorably. Fibrositis cases, such as muscular rheumatism, bursitis, neuralgias and periarthritis experienced good pain-relieving effects.

These clinical results substantiated impressions observed when this drug was used in animals after its synthesis by the Geigy Laboratories. The animal studies revealed Butazolidin to have usefulness in the relief of painful musculoskeletal disorders. Strong analgesic and antipryretic properties were noted, as well as an "anti-inflammatory" effect. The "anti-inflammatory" effects were delay and reduction of local tissue reaction produced by chemical and physical irritants.

The manufacturer reports a high degree of drug tolerance, as shown by toxicity studies in animals. Butazolidin, first used in Europe, was found quite efficacious in relieving rheumatic conditions as outlined. In the United States, Butazolidin has been subjected to intensive clinical investigations approximately two years. Papers read at the American Rheumatism Association meeting indicated that Butazolidin can produce prompt and prolonged relief from pain. On pain relief, musculo-skeletal tissue mobility is increased. Spasm is reduced. Dimunition of swelling also occurs in some cases.

A majority of the cases treated reacted favorably. Fortunately, its good effects are manifest within a period of two to four days. If a patient does not experience relief within a week, Butazolidin therapy should be discontinued. To use larger therapeutic doses where improvement is not thus observed carries a serious risk of producing toxic side reactions. When maximum relief has occurred, Butazolidin dosage reduction to minimally effective amounts is indicated. Such therapy procedure minimizes occurrence of side reactions; for one consciously accepts a serious hazard when using this drug. Furthermore, there is no proof that it has curative values. Thus far, medical reports do not make such claims.

We have been using Butazolidin since the reports were made at the American Rheumatism Association convention. Very close supervision is maintained over these cases. Laboratory tests are made at frequent intervals. Thus, reactions are promptly detected, and medication is immediately interrupted. Use of this drug without close personal supervision, plus adequate laboratory checkup is not warranted. Danger of serious side reactions occurring, with grave results to the welfare of the patient may otherwise results. Butazolidin should be used only in cases that do not respond to present tried and proven treatment procedures. As yet,

Butazolidin is simply one more weapon in rheumatologic therapy.

Serious side reactions to be carefully watched for in Butazolidin medication are:

1. DRUG RASH: A very few of the patients experienced a drug rash. Medication interruption produced its disappearance.
2. NAUSEA: The drug is best given on a full stomach. Some can take Butazolidin without nausea, even on an empty stomach. If nausea appears, an antacid will help, such as magnesium hydroxide, or calcium carbonate.
3. EDEMA: Edema has occurred in two of our cases thus far. It cleared quickly upon withdrawal of Butazolidin.
4. SODIUM: Salt intake should be restricted. Butazolidin tends to cause sodium retention, with resulting localized or generalized swelling. It should not be used in patients with cardio-vascular, renal or hepatic difficulties.
5. PEPTIC ULCER: As yet, no patient has reacted with peptic ulcer activation. The medication should not be used in individuals with active peptic ulcer or with duodenal ulcer history.
6. ABDOMINAL CRAMPS: Marked abdominal cramping may occur in patients taking Butazolidin. Persistent abdominal cramping may necessitate addition of a non-sodium-containing antacid. Butazolidin discontinuance may become necessary. The patient may stop the drug because of cramping.
7. AGRANULOCYTOSIS: In our experience agranulocytosis, thus far, has not appeared in those on Butazolidin therapy. A few cases have reacted with a mildly-moderate fall in the neutrophile count. Temporary elevation of the white blood cell count was noted in three cases. The particular chemical structure of this drug requires frequent white and differential cell counts. Any indication that the granulocyte count is being definitely reduced, calls for immediate Butazolidin interruption. *In some personal communications I have received, three severe agranulocytosis cases have been observed.* They had not responded adequately to the drug withdrawal at the time the communications were written.
8. CHEMISTRY: The chemical structure of Butazolidin is such that in due time, if the medication is used without proper supervision, bone marrow toxic effects may result. It should be given with extreme caution.
9. SECONDARY ANEMIA: In our experience a troublesome factor has been a secondary anemia production. There develops a definite reduction in the red cell count and hemoglobin determination. This has not been sufficiently emphasized. It points up the urgent need for frequent red and white cell counts. Laboratory checkup tests are done weekly the first six weeks of treatment; thereafter, every ten days; and later twice a month.

Butazolidin therapy, for best effects, should not be used persistently for weeks or months, but over a period of days. Intervals of non-treatment should alternate with Butazolidin treatment periods.

When a patient has not derived benefit from Butazolidin therapy in four to seven days, it should be discontinued. He who does not get good results from it should not be pushed to larger and larger dosages. It should be promptly stopped.

SUMMARY

Butazolidin (Phenylbutazone) is still in process of being evaluated as a therapeutic drug in rheumatic disorders. Inasmuch as serious side reactions may occur, it should be given only under the close personal supervision of the physician. Frequent laboratory examinations of the red and white blood cells are imperative.

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PERITONEAL BANDS OF NON-SURGICAL ORIGIN

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Since Virchow's[1] comprehensive report, published in 1853, the frequent occurrence of intraperitoneal bands has been noticed by all pathologists and by most surgeons. Harris,[4] writing in 1914 on duodenal obstructions, apparently was the first in this country to notice the problem of abnormal peritoneal bands. Following this treatise there were a number of articles published which were primarily concerned with the entity titled "gastro-intestinal stasis." This condition appears to have been quite fashionable in surgical circles during the first two decades of the present century, enjoying considerable attention from such leading men of that time as C. J. Mayo, the elder Coffey, Arbuthnot Lane, Franklin Martin *et al.* Suffice it to say that these observations have become of interest mainly from a historical standpoint. In more recent years there has been a resurgence of interest in certain types of peritoneal bands, particularly the hepatoduodenocolic band of Harris, and excellent papers regarding this anomaly have been published by Solem,[5] Taylor,[6] Kantor[7] and Mahoney.[8]

Bryant[2,3] who has made the most accurate and valuable observations in modern literature concluded that the frequency of peritoneal bands has been greatly under-estimated. In his series of cases he found bands present in 91 per cent of the males, which included 18 fetuses. He also observed the same frequency of involvement of the various types of bands in both fetuses and adults. This finding seemed to confirm the development origin of these bands or "adhesions." His findings are summarized as follows: 1. The transverse colon is more frequently involved by adhesions than any other abdominal viscus. 2. Within a range of variation not usually exceeding 10 per cent, the rate of involvement of a given viscus may be greater in the male or in the female, and this relative rate of involvement in regard to sex may further vary with age. 3. The rate of involvement by adhesions, is for several viscera higher in the fetus than at later ages, as for the transverse colon in the male and the terminal ileum in the female. 4. The rate of involvement by adhesions increases rapidly with progressive age for certain other viscera, as for the sigmoid flexure in the male and the adnexa in the female. 5. The age of 40 is critical in both sexes with reference to the average number of viscera involved by adhesions in any given case. After the age of 40 there is a sudden increase of involvement by about 50 per cent in both sexes, the increases being somewhat more marked in the male than in the female. 6. Complexity is practically a synonym for age, with regard for the number of viscera involved in any given adhesive process. This increase in complexity amounts to over 200 per cent after forty years of age. 7. Variety in the character of the adhesions present also increases with age. A sudden marked increase of nearly 100 per cent occurs in the 30-40 year decade, or 10 years earlier than the onset of the marked increase with regard to the average number of viscera involved, and the onset of the increase in the complexity of the adhesions themselves. 8. The distinguishing characteristics of congenital or developmental adhesions, are simplicity and lack of variety in type. 9. The distinguishing characteristics of acquired adhesions, are complexity and variety in type.

CASE REPORTS

Case 1: Mrs. E. P., white, age 50, was admitted to the hospital complaining of agonizing paroxysms of abdominal colic. The preceding night she had developed some mild abdominal pain which she attributed to an

FIG. 1. (*Case* 1.). *The genito-mesenteric fold of Reid is seen passing over the terminal ileum and cecum and extending upward toward the duodenum.*

"upset stomach." During the night this progressed to become severe paroxysms of cramping located in the para-umbilical and left suprapubic regions. She had little or no nausea but tried to induce vomiting in the hope that this might bring relief. Her bowels had always been regular and she had never noticed any abnormal stools. By dieting she had maintained her weight at around 100 pounds. She had had two normal full-term pregnancies. She had had no other illness than the usual ones of childhood. There was no history of previous operations or of injury to the abdomen. A complete review of the body systems was negative except as indicated above.

The physical examination was negative except for the abdomen. This was very slightly distended. There were no operative scars present. The recti were not rigid and no masses or enlarged organs could be felt. Diffuse tenderness was present throughout the abdomen but was most marked in the suprapelvic area and in the right lower quadrant. Slight rebound tenderness was present in the latter area. Auscultation revealed peristaltic rushes and increased borborygmi. When the finger was inserted deeply into the rectum a tender mass could be felt. Vaginal examination was negative except that a second degree retroflexion was present and manipulation of the cervix caused reproduction of the primary pain. A scout film of the abdomen gave no useful information and the routine laboratory examinations were normal except for a leucocytosis of 12,500 of which 88 per cent was polymorphonuclear cells.

The diagnosis was acute intestinal obstruction, possibly due to volvulus of the small intestine, and laparotomy was performed under spinal anesthesia about 20 hours after the onset of this patient's illness.

Through a left para-median incision the peritoneal cavity was opened. A large quantity of sero-sanguinous fluid welled out of the opening and the small intestine was moderately distended. A loop of greatly distended and dark plum-colored ileum was seen in the depth of the pelvis. Further inspection showed a mesenteric band extending from the root of the iliac mesentery to the posterior surface of the right utero-sacral ligament where it merged with the lower uterine segment (Figure 1.). This was completely obstructing a loop of ileum approximately 20 cm. long at a level about 15 cm.

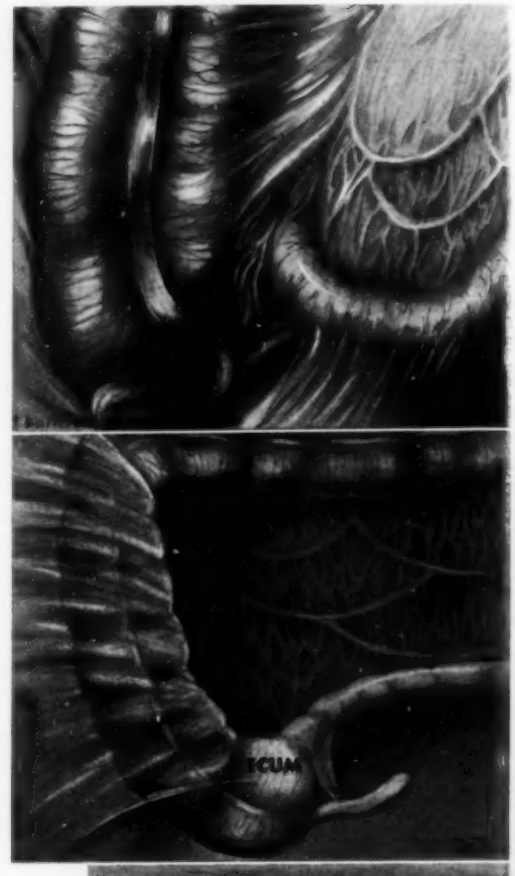

FIGURE 2. *(Case 2.). The ileal membrane, or Lane's Fold.*

FIGURE 3. *Jackson's Veil, or pericolic band.*

FIGURE 4. *The cholecystoduodenocolic membrane. The anterior liver edge is retracted upward to show the important relationships of the band.*

above the ileocecal junction. This constricting band was severed and the strangulated portion of bowel was packed for 25 minutes with very hot wet laparotomy pads. The color of the bowel segment improved markedly, so, after removing a normal appendix, the abdomen was closed without drainage.

The patient made an uneventful recovery and was dismissed from the hospital on her eighth post-operative day. At the time of this writing, three years later she had had no further difficulty.

Case 2: Mrs. J. McK., white, aged 52, was admitted to the hospital complaining of abdominal pain, nausea and vomiting. Five days prior to admission she had eaten some fried cabbage which was vomited after a few hours. The following day she developed some mild cramping pains in the abdomen. An enema produced a fairly copious bowel movement but the pain gradually became worse. Three days later she was first examined and at this time she was vomiting incessantly and had had no bowel movements. The abdomen was diffusely moderately enlarged and was tender throughout. No masses could be felt superficially or by vagina or rectal examination. X-ray following a barium enema revealed an obstruction of the small bowel with a normal colon. Laboratory examination was normal except for a leucocytosis of 14,000, of which 84 per cent were polymorphonuclear cells.

The diagnosis of small bowel obstruction, possibly due to a band, was made, and laparotomy was advised but the patient refused to permit surgery. A Cantor tube was introduced and intravenous feeding was begun. There was no change in the condition of the patient and three days after admission to the hospital she agreed to submit to an operation. This was one week after the onset of her illness.

Under pontocaine-sodium pentothal anesthesia the abdominal cavity was entered through a right paramedian incision. A moderate amount of sero-sanguinous fluid was encountered and an obstruction of the ileum was found about five cm. above the ileo-cecal valve. This was seen to be due to a congenital fold of peritoneum which, extending from the right lateral parietal wall beneath the cecum and crossing the terminal ileum, merged into the upper surface of the ileal mesentery. This four cm. band, which blended in to the serosa of the ileum, was divided to free the bowel. A retro-peritoneal appendix was removed and the abdomen closed without drainage.

The postoperative course was complicated by a central pneumonia which subsided rapidly under appropriate treatment. The patient was discharged from the hospital on her twelfth postoperative day and has enjoyed good health up to the date of this writing over three years later.

DISCUSSION

There are seven distinct types of intraperitoneal accessory membranes or bands.

1. *The Genito-mesenteric Fold.* (Reid's Band). This membrane is a triangulae fold of peritoneum which originates on the back of the terminal mesentery from the junction of the second and third parts of the duodenum to the region of the suspensory ligament of the ovary or testis. It is on the right side and is often closely related to the uterus, right ovary and appendix. It is said to be the cause of binding down and kinking of the appendix. The first case is thought to be an example of this condition.

2. *The Ileal Membrane.* (Lane's Fold.) This extends from the right iliac fossa to the anti-mesenteric border of the terminal five to eight cm. of the ileum. It is a prolongation of the parietal peritoneum of the right iliac fossa. Lane postulated that shortening of this band leads to obstruction of the terminal ileum at the point of fusion of the band. This theory is confirmed by the circumstances related in the second case.

3. *The Pericolic Band.* (Jackson's Veil). This band is commonly found in operation for appendicitis. It is a thin delicate fold of peritoneum which extends from the right posterior parietal wall lateral to the ascending colon to the anterior taenia of the cecum of lower portion of the colon.

4. *The Cholecystoduondenocolic Membrane.* (Cysto-gastro-colic Band). Niles'[9] has suggested the term Harris' Band for this not uncommon anomaly although it was first noticed by Virchow in 1853. It is a variable of development of the hepato-gastric ligament and usually stretches from the under edge of the liver, over the gall bladder across the duodenum (to which it may be fused) to the right transverse colon. Mahoney[8] points out that the wide anatomic variations of this membrane may cause symptoms referable to any of the viscera to which it happens to be attached.

FIGURE 5. *The mesosigmoid membrane.*

FIGURE 6. *The mesocolicojejunal band of Coffey exposed by turning the transverse colon upward.*

5. *The Mesosigmoid Membrane.* (Lane's First-and-Last Kink). This band is the left sided counterpart to Lane's ileal band and is a hypertrophic shortened duplication of the peritoneum of the left iliac fossa binding the sigmoid colon down to the brim of the pelvis. According to McGregor [11] it was the first to appear embryologically and last in the sense that it involves the last or lowermost part of the bowel.

6. *The Mesocolicojejunal Membrane.* (Coffey's Band). This is a rare band of variable width and length extending from the posterior surface of the transverse mesocolon to the antimesenteric border of the jejunum below its point of emergence past the ligament of Treitz.

7. *The Intercolic Membranes.* (Payr's Membranes). These are folds of the colic peritoneum which are sometimes seen extending across the acute angles of the hepatic and splenic flexures of the colon. They are of little or no clinical significance although it was once thought that they contributed to stasis of the fecal flow in the colon.

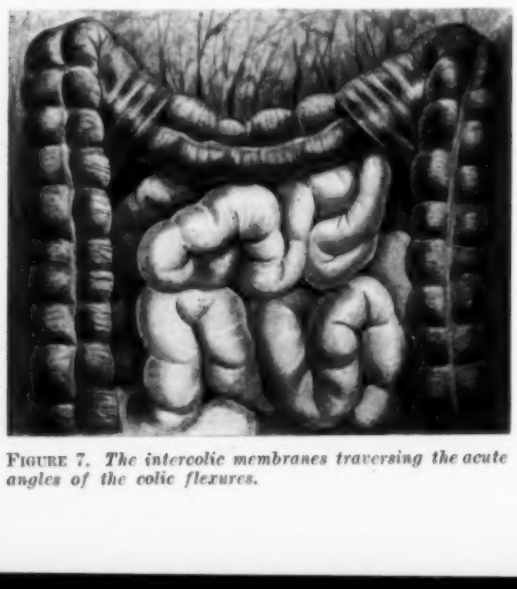

FIGURE 7. *The intercolic membranes traversing the acute angles of the colic flexures.*

CONCLUSION

There is very little information available in the medical literature regarding the occurrence of congenital intra-abdominal bands. A few scattered reports have called attention to the presence of these dangerous structures but many surgeons are not familiar with the various types of bands which may be present within the scarless abdomen. For this reason the seven clinically recognizable kinds of congenital bands have been described and two cases have been presented to illustrate the presence of potentially lethal congenital bands.

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PANCREATIC CYSTS

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Four patients with a cyst of the pancreas have been studied and treated at this hospital within the past two years. In the previous 10 years no similar cases could be found. It is the purpose of this paper to discuss pancreatic cysts in general, to emphasize the roentgenological signs associated with them, and to present the four cases which stimulated this work.

CLASSIFICATION

The classification used by most writers was originated by Mahorner and Matson[20] in 1931. This was slightly modified in 1946 by Adams and Nishijima[1], and this modification is the one closely followed here.

1. *Retention Cysts* — These unilocular cystic lesions result from obstruction to the outflow of pancreatic secretions by either calculi, tumor or chronic pancreatitis. They are usually lined by epithelium similar to that lining the pancreatic ducts. These cysts are apt to be small and are usually not clinically significant. Actually, the clinical impression that retention cysts result secondary to duct obstruction has practically no experimental background. In laboratory animals one finds fibrosis of the parenchyma, atrophy of the acini, and duct dilatation when the ducts are obstructed. Lazarus, as quoted by Meyer[21], was able to produce a small retention cyst after simultaneously ligating the main duct and injecting tincture of iodine into the substance of the gland. Confirmation of this work could not be found.

2. *Proliferative Cysts*: These lesions are true neoplasms and histologically are either cystadenoma or cystadenocarcinoma. Both lesions are almost always multilocular. There is no associated history of trauma or of biliary disease.

3. *Embryonal Cyst*: These also are neoplastic in origin and histologically are either dermoids or teratomas.

4. *Parasitic Cysts*: The hydatid cyst of the pancreas is rare. Only 40 had been reported up to 1920. The cysticercus cellulosae may produce pancreatic cysts, but these are usually very small, being only seven to 15 mm. in diameter.

5. *Congenital Cysts*: This group includes those due to defective development. The majority are in the recently recognized entity of cystic fibrosis of the pancreas. The cysts are usually multiple and are frequently associated with congenital anomalies of other viscera, especially polycystic lesions in the kidneys.

6. *Pseudocysts*: This group includes (a) those collections of fluid which form within the substance of the pancreas as a result of a hemorrhage, necrosis, and other degenerative changes affecting the parenchyma of the gland, and (b) those collections of fluid which lie adjacent the pancreas and are due to the corrosive pancreatic ferments. These peripancreatic encysted fluid collections lie in the lesser peritoneal cavity. The degenerative cysts which arise within the gland will rupture into the lesser peritoneal cavity when the necrotic process reaches the anterior surface of the pancreas. If the foramen of Winslow is sealed off, the lesser omental sac becomes a closed space and the exudate becomes encapsulated by the proliferation of fibrous connective tissue.

The walls of these pseudocysts are dense connective tissue which will be unusually vascular if the collection of fluid is of relatively recent origin. There is no epithelial lining. This absence of an epithelial lining has been used to differentiate them from true pancreatic cysts of the retention or proliferative types. Differentiation is often impossible since a large retention cyst may have its epithelial lining undergo pressure necrosis, or the proliferative cysts may have their linings destroyed by pancreatic enzymes.

Several factors may account for the origin of pseudocysts. Trauma is undoubtedly an important one. The first result of trauma to the pancreas is hemorrhage. After this comes necrosis and the changes due to liberated pancreatic enzymes. These ferments may so alter the hemorrhagic fluid that it will be clear or straw colored, but it is more often dark brown or brownish green. The fluid may contain particles of sequestrated pancreatic tissue. It is intriguing that the pancreas is the only organ in the body which reacts to trauma by self destruction. Its anatomical position anterior to the vertebral column makes it unusually vulnerable to blunt trauma. Yet the part that trauma plays in pancreatic and peripancreatic accumulations of fluid has probably been ac-

FIG. 1. CASE 1. *There is a general widening of the duodenal loop with pressure on the inferior portions of the pyloric antrum and duodenal cap. The mucosal pattern in the descending duodenum is smoothed.*

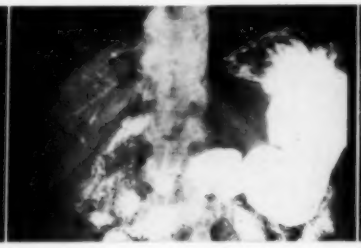

FIG. 2, CASE 1. *Examination done six months after the gastrojejunostomy and choledochojejunostomy. The cyst had been opened, drained and closed. There is no evidence of widening of the duodenal loop. The common duct is moderately dilated, and the liver radicals are well outlined with barium.*

FIG. C, CASE 2. *The body of the stomach is displaced forward and upward and stretched across a large soft tissue mass behind it. The duodenal cap is displaced to the right, and the transverse duodenum is displaced downwards. (Retouch of original film)*

corded undue significance in the early literature.[8,24,25,33] Acute and chronic pancreatitis accounts for many cases of pseudocyst, and some writers think the majority are due to such an origin. It is outside the scope of this discussion to consider the relationship of biliary tract disease to acute pancreatitis. Statistically, however, Pinkham[25] reported 10 cases of pseudocyst, six of which were thought to be secondary to acute pancreatitis and hemorrhagic necrosis. In four of these six there was associated biliary tract disease. Meyer[21] reported 31 cases and found cholecystitis and pancreatitis as the etiological agents in six (16.1 per cent). Adams and Nishijima[1] reported cholelithiasis in two (40 per cent) of their five cases. Judd[17] reported 17 (36 per cent) out of 47 cases had associated biliary tract disease. An additional factor which may produce a pseudocyst of the pancreas was reported by Russ[30] in 1921. His patient developed a cyst during the course of puerperal sepsis, and the possibility of an embolus, either sterile or infected, producing a necrotic area in the pancreas was suggested.

INCIDENCE

Cysts of the pancreas are rare. Adams and Nishijima[1] reported nine cases at the Lahey Clinic between 1926 and 1945. Warren[33] reported seven additional cases from the same institution between 1945 and 1948. Holt[14] was able to find 13 patients with a confirmed diagnosis of pancreatic cyst at the University of Michigan Hospital over an 11 year period. Rabinovitch and Pines[29] reported 17 cases in 1942. Matson and Mahorner[20] reported 88 cases in 1931 from the Mayo Clinic in a total of 723,797 admissions for an incidence of 0.012 per cent. Johnson and Gibson[16] could find only seven pancreatic cysts treated at the University of Kansas Medical Center in a 12 year period between 1936 and 1948.

Of the surgically important lesions, the pseudocysts are statistically much more prominent than any other group. Unfortunately, it is sometimes impossible to distinguish the retention and proliferative cysts from the pseudocyst due to occasional occurrence of necrosis of the epithelium lining the true cysts. This fact may cast some doubt on the accuracy of some of the surveys published. An extensive work on pseudocysts was done recently by Meyer[21] in 1949 when he was able to collect 31 cases of pseudocysts operated upon at Cook County Hospital between 1930 and 1948. Rabinovitch and Pines[29] reported 11 pseudocysts in a group of 17 pancreatic cysts. Waltman and Cleveland[32] reported on 55 cases of pancreatic cyst which had pathologic confirmation and classification. Twenty-four (43.6 per cent) of these were pseudocysts, 13 (23.6 per cent were retention cysts, seven (12.7 per cent) were cystadenomas, and four (7.2 per cent) were cystadenocarcinomas. In Warren's[33] 16 cases, eight (50 per cent) were pseudocysts, two were papillary cystadenomas, four were retention cysts, and one was a hemorrhagic cyst.

Proliferative pancreatic cysts are quite rare. Brunschwig[4] estimated that only about 40 cases of cystadenoma had appeared in the literature up to 1942. Haukohl and Melamed[12] were able to collect 11 additional cases between 1942 and 1950. Bowers, Lord and McSwain[3] in 1942 reported five cases of cystadenoma which comprised all the surgically treated cystadenomas at New York Hospital up to that time. These authors mentioned that four of these five cases had associated biliary tract disease but questioned any pathogenic relationship between the two.

In Johnson and Gibson's[16] seven cases, only one cystadenoma was found.

Pseudocysts occur about equally between the two sexes. The increased incidence of biliary tract disease in the female is probably offset by the increased occurrence of upper abdominal trauma in the male. Cystadenomas are much more common in the female. Haukohl and Melamed[12] found 89 per cent of the recorded cases were in female patients.

The cystic lesions of the pancreas are seen more commonly in the middle age group. They have been found in early life. Telling and Dobson[21] reported a pancreatic cyst in an 11 month old child. Eha[7] reported a congenital cyst of the pancreas without an epithelial lining in a five month old infant. The cases of cystic fibrosis of the pancreas are almost all seen in the first few months of life.

SYMPTOMS

The symptoms due to a pancreatic cyst are dependent upon the precipitating factors, the size and location of the lesion, and the duration of the disease. In many instances there are no subjective complaints since these cysts may be quite slow and insidious in onset. When the cyst is of traumatic origin, this fact is usually prominent in the history. In Meyer's[19] series the time interval between the traumatic incident and the discovery of the pseudocyst averaged five months. There is a triad of symptoms commonly found: Upper abdominal pain, pressure symptoms from adjacent organs, and the presence of a palpable mass.

Pain is the most constant and most important subjective complaint. It varies from a sensation of fullness after meals to sharp abdominal cramps. Its location is usually epigastric, but will often radiate through to the back. The pain, however, is apt to be quite variable in degree. Its location is of little diagnostic value. Weight loss, anorexia, nausea and vomiting are often associated. The occasional occurrence of chronic pancreatitis with a benign pancreatic cyst will add additional symptoms. These patients will have a history of long standing intermittent episodes of abdominal pain associated with the gradual development of an upper abdominal mass.

Diarrhea is seldom encountered. Jaundice may occur when a cyst has grown large enough to obstruct the common bile duct. Pressure on the portal vein may produce ascites. Only rarely does one find foamy stools, but an excess amount of fat in the stools may be a manifestation of pancreatic deficiency. Recently, Gross, Null and Loeb[10] reported a case of hydrothorax associated with pseudocyst of the pancreas. They were able to collect five other such cases from the literature. This has been likened to the formation of hydrothorax with a benign intrapelvic tumor in the female as seen in Meig's syndrome.

The presence of an abdominal mass is sometimes the only complaint. In most of these patients the history is of slow growth over a long period. The mass may enlarge suddenly. A phenomenon which is almost limited to pancreatic cyst is its spontaneous disappearance followed by re-appearance in a few days. According to Hess and Rutte[15] this occurs in about five-10 per cent of the cases. This unique behavior has not been adequately explained, but the most logical explanation is that emptying occurs through the duct of Wirsung when the pressure within the cyst has reached a certain level.

The abdominal mass is smooth to palpation, and often is so firm that fluctuation cannot be demonstrated. When the mass is located in the head of the pancreas, it is relatively fixed, but the tumor may descend slightly upon deep inspiration. When the mass is located in the tail of the pancreas, it may be slightly or even freely movable. Cysts which are of inflammatory origin are likely to be somewhat tender.

Laboratory data may be of some assistance in diagnosis. Pinkham[25] noted an elevated preoperative serum amylase in six patients which dropped promptly after drainage in four. He thought that a sustained elevation of serum amylase after acute pancreatitis suggested the development of a pseudocyst. Meyer[21] had preoperative serum amylase levels in eight patients, and found two of these abnormally elevated. In both cases a ruptured cyst was found at operation. There seems to be a slight increase in the incidence of diabetes in patients with pancreatic cyst. Warren[23] found diabetes in three (18 per cent) of 16 cases. Bowers et al[2] found it in three of five patients and reported postoperative improvement in one. On the other hand, no patient in the 31 cases of Meyer had diabetes. The stools should be examined for free fat, and if duodenal contents are obtained a deficiency of pancreatic ferments may be found.

ROENTGEN MANIFESTATIONS

Cystic lesions of the pancreas cannot oft-

en be visualized directly on the scout films of the abdomen since their density is approximately the same as that of the surrounding tissue. Occasionally, a soft tissue mass can be clearly outlined and rarely there will be calcifications in the walls of the cyst. Haukohl and Melamed[12] reported two cystadenomas with stromal calcification but were unable to find similar reports. Pascucci[23] found only 16 cases of pseudocyst of the pancreas which had an associated pancreatic parenchymal lithiasis. Alexander and Lowder[2] reported two patients who had pancreatic calcifications arranged in a spherical fashion. A cystic change within the pancreas was accurately predicted.

It is unfortunate that there is no excretory method of visualizing the pancreatic ducts. X-ray findings are dependent upon the site of origin of the cyst and its subsequent direction of growth. Diagnosis depends upon indirect evidence obtained by outlining contiguous structures.

Cysts in the head of the pancreas cause a smooth, rounded enlargement or fanning-out of the duodenal loop. This pressure will alter the mesial ends of the duodenal mucosal folds. It must be noted that hypersthenic and obese individuals with high, horizontal stomachs normally present large duodenal loops which may be mistakenly called abnormal widening. The duodenal loop is displaced to the right, often anteriorly, and may show a slight narrowing of the lumen. This diminished diameter of the duodenum may be enough to produce partial obstruction with delayed gastric emptying. The pyloric end of the stomach is usually not deformed or displaced. The transverse colon is displaced downwards and forward.

Cystic lesions arising in the body or the tail of the pancreas displace the body of the stomach forward, and it may be either upwards or downwards. There is usually a concave deformity of the stomach, the concavity being towards the tumor. This defect is located on the lesser curvature of the stomach when the lesion arises from the head or body of the pancreas. The indentation is on the greater curvature of the stomach when the cyst arises in the tail. In the latter situation, it may entirely disappear in the erect position. A tumor extrinsic to the stomach can sometimes be pushed away from the stomach by palpation under the fluoroscope, with alterations of the deformity. The margins of the gastric defect are apt to be fuzzy and indistinct when produced by an extrinsic mass. Also this compression deformity may show a striking change in form when the patient is changed in position. The mid-transverse colon is displaced forwards, and usually downward. There may be compression and partial obstruction. Sometimes the distal transverse duodenum is displaced downwards and in such a case the duodenum proximal to this may be a little dilated. Other signs of significance than those mentioned are alteration of the left psoas shadow and compression of the left ureter causing a partial obstruction. Sometimes the distal transverse duodenum is displaced downwards and in such a case the duodenum proximal to this may be a little dilated. Other signs of less significance than those mentioned are alteration of the left psoas shadow and compression of the left ureter causing a partial obstruction.

SURGICAL MANAGEMENT

There are three methods of attack open to the surgeon confronted with a cyst of the pancreas: (a) Marsupialization, (b) total excision, or (c) internal drainage. Each method has some merit and certain limitations.

Marsupialization or external drainage is the simplest method with the lowest mortality and is the one most commonly used. It seems to be the elective procedure in critically ill patients. It is also the method of choice in the presence of severe degrees of pancreatitis or peripancreatitis where resection of the cyst is impossible or unwise. Unfortunately, marsupialization is sometimes followed by recurrent cyst formation[26,27] or by a persistent external fistula.[21] A persistent fistula may have frequent hemorrhages, may become secondarily infected, or may cause considerable inanition by continual loss of pancreatic juice. The continued drainage of these pancreatic ferments may also cause a painful erosion about the abdominal wall stoma. It has been demonstrated in the past that the fistulas can sometimes be eliminated by chemical irritants, such as sodium morrhuate. External irradiation has been used successfully in a few patients to destroy the epithelial lining of the tract and produce fibrotic closure. A second operation is occasionally done where the fistulous tract is anastomosed to the jejunum. Banthine will inhibit the external secretions of the pancreas and has been used with gratifying results[27] in postoperative fistulas. Warren[33] states that marsupialization should be con-

fined to pseudocysts of the pancreas, and that external drainage should be done before the cyst walls become thick and rigid.

Total excision should be done in all cases where the cyst can be easily enucleated. This requires careful and patient dissection. Retention cysts are the ones most easily extirpated because they have a paucity of inflammatory reaction around them. Ideally, all proliferative cysts should be removed completely because these may undergo malignant degeneration. Gurwitz and Hurwitz[9] feel that non-proliferative cysts in the head of the pancreas should not be extirpated because of the high incidence of injury to the common bile duct and to the pancreatic duct. Kunc[19] warns that complete removal is technically arduous and is apt to be accompanied by profuse hemorrhage. In two European series there was a mortality rate of 10.7 per cent which is said to increase to 55 per cent if the intended extirpation of the cyst proves impractical during the operation. Total excision is usually reserved for the smaller cysts which have few or no adhesions about them. Even then, trauma to the pancreas during surgery may produce an acute pancreatitis or a pancreatic fistula.

Internal drainage is done by anastomosis of the cyst with the stomach, gallbladder, duodenum or jejunum. This has become increasingly popular in recent years. This procedure was first done in 1927 when Hahn[21] anastomosed a cyst to the jejunum. There were early objections to this method since it was felt there was a danger of spilling alimentary tract contents into the cyst cavity producing more necrosis of the pancreas. This, however, has not occurred, apparently because there is no connection between the cysts and the main pancreatic ducts. Cystocholecystostomy seems to be the least desirable since the cyst is prone to empty incompletely, and since there is usually associated biliary tract disease. When internal drainage is done, anastomosis to the jejunum seems preferable with or without utilization of the Roux-Y principle.[22]

Pancreatic cysts should not be aspirated. Such a procedure is futile and dangerous. A chemical peritonitis can easily be produced.

Koucky, Beck and Todd[18] have called attention to cases of pseudocysts which rupture before operation. These authors reported six such cases from Cook County Hospital. The perforative episode was quite similar in all, and closely resembled a patient with a perforated peptic ulcer. These acutely ill patients also resembled those with a ruptured gallbladder. A history of an upper abdominal mass which disappeared with the onset of the symptoms of peritonitis helps in the differential diagnosis. Four of these six patients died.

REPORT OF CASES

Case 1. This 65 year old, white female, first entered the hospital on 11/26/46, complaining of a continuous mild jaundice of four-five months duration. The icteric index varied between 10.6 and 18.5. The cephalin flocculation test was negative. No abdominal masses could be felt, and the liver was not enlarged. The gallbladder failed to concentrate Priodax tablets. The jaundice gradually cleared in a couple of weeks, and she was well for about 15 months. Her second admission was on 2/17/48, with a history of progressive jaundice for one month and an icteric index of 31.7. Laparotomy was done 2/21/48. The gallbladder wall was thickened without stones, and the cystic and common ducts were moderately dilated. A probe could be easily passed from the common duct into the jejunum. The head of the pancreas was enlarged but not hard. The gallbladder was removed, a "T" tube placed in the common duct, and the head of the pancreas biopsied. Microscopically, the gallbladder showed chronic cholecystitis; the pancreas was reported as showing recent hemorrhage due to surgery. Her postoperative course was smooth, and the "T" tube was removed after six weeks.

The third admission was on 10/19/51, with a history of progressive jaundice for two and one-half months. Her icteric index was 149.2. The total serum proteins were 6.6 gms. per cent. A gastro-intestinal series showed some fixation of the distal 3 cm. of the stomach without displacement. The duodenal cap was elongated, and the duodenal loop was moderately widened. A pre-operative diagnosis of tumor of the head of the pancreas was made. Laparotomy was done on 10/23/51. A multiloculated cystic mass 4 x 6 cm. was found in the head of the pancreas which contained a light-greenish colored fluid. This cyst was obstructing the lower end of the common bile duct. A generous biopsy of the pancreatic wall was done. The opening into the cyst was closed after aspiration of the cyst contents. An anterior gastrojejunostomy and a choledochojejunostomy were performed. Due to this patient's poor condition it was not thought advisable to attempt extirpation of the tumor. Amylase content of the cyst fluid was 20 mg.

Postoperatively she had a stormy course, developing hypokalemia and cystitis. She was markedly improved when discharged 11/17/51. Pathologic diagnosis was benign cystadenoma of the pancreas. She was admitted a fourth time on 12/5/51, with a history of severe vomiting of 36 hours duration which had begun with a chill and a left-sided convulsion. She had weakness of the left arm and leg. All symptoms and signs cleared within 48 hours on parenteral fluids. She was last seen on 4/23/52, and had no complaints. No abdominal mass could be palpated. A gastro-intestinal series done at this time showed no widening of the duodenal loop.

Case 2: This 74 year old, white male, first entered the hospital 10/9/50, with a history of intermittent abdominal pain and distention for a period of two years. A large upper abdominal mass had been noted for about four-five months by the patient and his physician. There had been no weight loss. A gastro-intestinal series showed the fundus of the stomach displaced upwards, the body of the stomach displaced forward, and the pyloric end of the stomach displaced forward and to the right by a large upper abdominal mass posterior to the stomach. There was no delay in emptying of the stomach. The transverse colon was stretched across this abdominal mass, being displaced anteriorly. The preoperative diagnosis was pancreatic cyst. The urine was negative for sugar. Laparotomy was done 10/14/50, and a large mass was found arising from the lesser peritoneal cavity, extending under and through the transverse mesocolon. This mass was a multiloculated cyst containing 4,000 cc. of brownish-green fluid. After aspirating this fluid, the cyst was excised. There was disruption of the abdominal wall on the ninth postoperative day. This was closed and no additional difficulty was encountered. Microscopically, the cyst cavity was lined by a dense hyalinized fibrous tissue without any definite epithelial lining. This was thought to be a retention cyst with necrosis of the lining. This patient has had no additional intestinal symptoms. A transurethral prostatectomy was done 8/16/51, with a normal postoperative recovery.

Case 3. This nine year old, white boy, fell from his bicycle on 4/21/51, striking his upper abdomen against the handlebar. He was admitted to his local hospital about an hour later with a blood pressure of 100/64, a pulse of 120. He was cold, sweaty, and his nail beds were cyanotic. A presumptive diagnosis of ruptured spleen was made. Following transfusions he did so well over the next 10 days that exploration was not thought necessary or advisable. One month after his initial injury he fell from a teeter totter and again was brought to the hospital with a clinical picture similar to that on previous admission. Within 24 hours, he was well again. During the next month a mass in the left upper abdomen appeared and grew gradually. He began having difficulty ingesting solid foods, but there was no vomiting. There was about 10 pounds weight loss in the two months following the original injury.

The patient was first seen at Wesley Hospital on 6/22/51. He was lethargic and seemed chronically ill. A large, firm smooth mass could be easily felt in the left upper abdomen which extended obliquely downwards to the umbilicus. This mass was fixed, and did not move with respiration. It was slightly tender. There was a mild anemia with 3.59 million RBC and 10.5 gm. per cent hemoglobin. Intravenous pyelo-

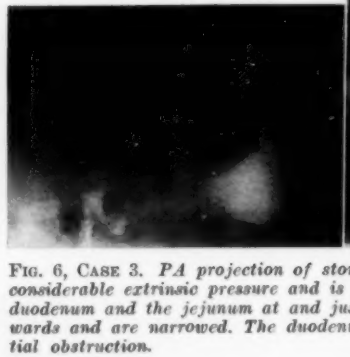

FIG. 4, CASE 2: *Lateral view of stomach and duodenum. The body of the stomach is displaced forward, and the distal one-third shows the mucosal pattern is "ironed-out." The duodenal cap is lying somewhat anteriorly.*

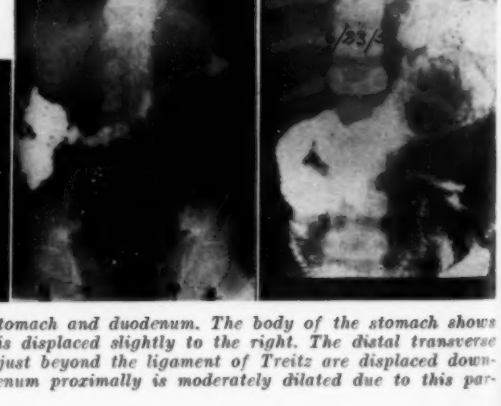

FIG. 5, CASE 2: *The transverse colon is stretched across an ill-defined but definite soft tissue mass in the upper abdomen.*

FIG. 6, CASE 3. *PA projection of stomach and duodenum. The body of the stomach shows considerable extrinsic pressure and is displaced slightly to the right. The distal transverse duodenum and the jejunum at and just beyond the ligament of Treitz are displaced downwards and are narrowed. The duodenum proximally is moderately dilated due to this partial obstruction.*

grams showed essentially normal kidneys. A gastro-intestinal series showed the stomach to be displaced anteriorly and slightly to the right by a large retroperitoneal mass. There was a deformity of the stomach which changed as the patient's position was altered. The transverse duodenum was irregularly narrowed and was displaced downwards. A preoperative diagnosis of pseudocyst of the pancreas was made. At operation on 6/26/51, a large, unilocular cystic mass was entered through the transverse mesocolon. Approximately 1,000 cc. of dark greenish fluid was aspirated. It was impossible to remove the cyst because the walls were not sufficiently developed. For this same reason internal drainage was not thought feasible. The cyst was marsupialized and the cavity packed open. Microscopic examination of a portion of the cyst showed a very vascular fibrous tissue. The amylase content of the cyst fluid was 196 mg. per cent. His postoperative course was smooth. The packs were removed slowly from the cavity over a six weeks' period. On 8/13/51, the cavity measured two by four by four cm. and a sinus tract about 2.5 cm. long led to the anterior abdominal wall. When last seen on 12/28/51, all drainage had cleared, and the patient had no complaints.

Case 4: This patient was a 71 year old white female whose only complaint was that of a feeling of fullness in her left abdomen which she had noticed for a couple of months. There was no nausea, vomiting or weight loss. On examination there was a firm, freely movable, non-tender mass in the left flank which was about the size of a large orange. This mass was easily separated from the left kidney by pyelographic study, and each kidney lumen was normal. The colon was not displaced. There was some extrinsic pressure on the greater curvature side of the stomach, but the wall of the stomach was not involved. The preoperative diagnosis was pancreatic cyst.

Laparotomy was done the eighth of August, 1952. The mass was located in the most lateral portion of the tail of the pancreas and was not adherent to any other intra-abdominal organ. It could be easily delivered through the abdominal incision. The lesion was totally excised without difficulty. The mass measured six by seven by 10 cm. It was nodular but well encapsulated. On cut section it seemed to be a solid tumor, yet presented a very loose stroma which surrounded tiny spaces filled with a firm, almost translucent, gelatinous material. Microscopically, these numerous, small spaces were separated by dense fibrous septa; the spaces were lined by orderly columnar epithelial cells. The diagnosis was benign cystadenoma of the tail of the pancreas. Her postoperative course was uneventful.

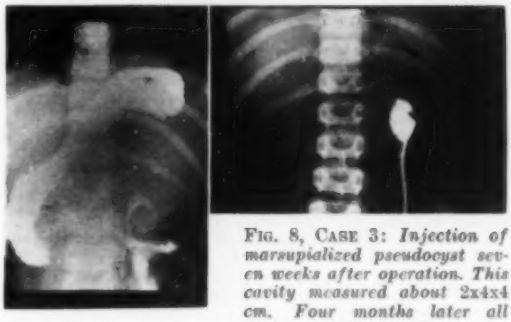

FIG. 8, CASE 3: *Injection of marsupialized pseudocyst seven weeks after operation. This cavity measured about 2x4x4 cm. Four months later all drainage had cleared.*

FIG. 7, CASE 3: *AP projection of stomach. Note the considerable difference in the pressure deformity of the stomach when position is changed from PA to AP. The left kidney lumen is still well outlined from previous intravenous urogram. There was no deformity of the kidney. The partial obstruction in the transverse duodenum is again shown.*

SUMMARY

1. Cystic lesions of the pancreas have been discussed according to their classification, incidence, symptomatology, roentgenological findings, and surgical management.

2. Four patients with a cyst of the pancreas have been presented: a cystadenoma of the head of the pancreas which produced obstruction to the common bile duct and jaundice, a retention cyst which was of considerable size, yet was successfully extirpated, a post-traumatic pseudocyst which was marsupialized, and a cystadenoma of the tail of the pancreas which caused only minimal extrinsic pressure changes on the greater curvature of the stomach.

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OBITUARIES

HENRY CLARENCE WEBER, M.D.
1882-1952

Henry C. Weber, M.D., long-time Bartlesville physician, died December 10, 1952.

Doctor Weber was a member of the State Board of Medical Examiners, a past president of the Oklahoma State Medical Association, member of the American College of Surgeons and a trustee of the Oklahoma Blue Cross and Blue Shield. He was a director in the Public Service Company of Oklahoma and a director of the Union National Bank of Bartlesville. He was a trustee in the Bartlesville Lodge 284 A. F. and A. M. and had been a member of the Bartlesville Chamber of Commerce since its organization. He was born in Dempseytown, Pennsylvania February 15, 1882.

Survivors include his widow of the home, one brother and one sister-in-law.

JOHN A. WRENN, M.D.
1908-1952

John A. Wrenn, M.D., Sulphur, died suddenly December 20.

Doctor Wrenn, who had practiced in Sulphur since 1951, was formerly a physician at the Veterans' hospital there and for a short time practiced in Ada. Doctor Wrenn was a graduate of the University of Missouri and the Washington University School of Medicine, St. Louis.

He is survived by his widow, Mrs. Maxine Wrenn, one daughter, and one brother. Burial was at Standsberry, Mo., his former home.

EDWARD D. RODDA, M.D.
1892-1952

E. D. Rodda, M.D., an Okmulgee physician for the past 28 years, died December 18 in a St. Louis hospital following an operation.

A native of Weir, Kansas, Doctor Rodda was a member of the Presbyterian church. He was a veteran of World War I and a member of both the American Legion and the VFW. In 1939 he served as president of the Okmulgee Gun Club and in 1946 he was elected chairman of the board of directors of the Okmulgee Country Club. He was active in all local medical activitoes. In 1931 he studied surgery in Vienna, Austria.

Survivors are his widow of the home, one son, one sister and two brothers.

ROBERT C. TAVLIN, M.D.
1920-1952

Robert C. Tavlin, M.D., Okeene, died suddenly at his home November 30. He had been ill with influenza for several days prior to his death but had recovered enough to resume work at the hospital a few days before his death. Before coming to Okeene in September, 1950, he practiced in Mooreland.

Detor Tavlin was born in Chicago March 28, 1920. He graduated from the University of Illinois Medical School in 1943 and interned at the Henrotin Hospital in Chicago. He served 26 months as a battalion medical officer in the army and then served a residency at Walther Memorial hospital in Chicago.

Survivors include his widow and four children, his parents, one brother and one sister.

WILLIAM G. McPHERSON, M.D.
1886-1952

William G. McPherson, M.D. died December 13 after a short illness. He had practiced medicine in Oklahoma since 1907. Before coming to Oklahoma City in 1943, where he practiced in Capitol Hill, he practiced at Ardmore.

Doctor McPherson was born at Harmony, Arkansas. He opened his first office in a small community between Wewoka and Holdenville and later practiced at Fort Towson, Healdton, County Line and Wynnewood before going to Ardmore.

Survivors include the widow of the home, two daughters, two brothers, and four sisters.

CLARENCE EDWARD LEE, M.D.
1875-1952

Clarence Edward Lee, M.D., Oklahoma City, died December 6.

The first superintendent of the old Oklahoma County Tuberculosis Hospital, Doctor Lee retired in 1949 and moved to California for a short time.

Doctor Lee was born in Frankfurt, Kentucky April 13, 1875. He was graduated from the University of Kansas Medical School, Kansas City, in 1901. He served with the medical corps in World War I and left the service with the rank of colonel. He was a member of the Methodist church, American Legion, Woodmen of the World, Shrine, Scottish Rite and was active in medical activities.

TINIA CAPITIS DUE TO TRICHOPHYTON SCHOENLEINI (FAVUS)

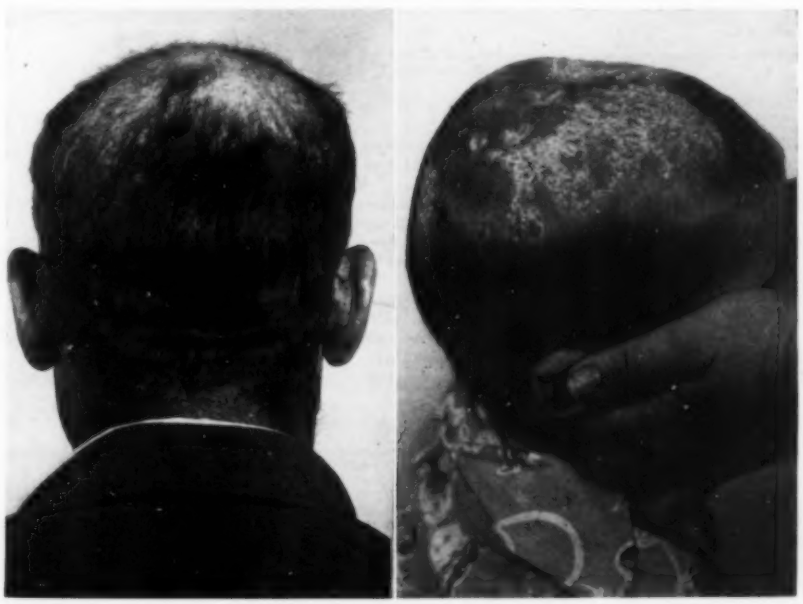
REPORT OF SIX CASES IN OKLAHOMA

PAUL O. SHACKLEFORD, M.D.
ROBERT J. MORGAN, M.D.
C. JACK YOUNG, M.D.
JOHN H. LAMB, M.D.

The term "favus" refers to a chronic fungus infection of the hairs of the scalp and, less commonly, to other areas of skin caused by *Trichophyton Schoenleini* (*Achorion schoenleini*). Characteristic yellowish, cup-shaped crusts known as scutula are produced on the scalp, with a peculiar mouse odor and frequent evidences of atrophy of the skin.

In reviewing the literature salient points are brought out by almost all of the writers, which we feel to be of interest in discussing this disease: 1) favus[1] does not involute at puberty to differ from the usual fungus infection of the scalp; 2) the disease occurs particularly in the poor and unclean, filth and neglect playing important roles; 3) it most commonly occurs in Poland, Russia, France, Egypt, the Balkans, and country about the Mediterranean basin; 4) it mainly is an anthrophilic rather than zoophilic infestation. Incidence of the disease in the United States is small and reports from the literature reveal scattered locations by states such as New York[2], North Carolina[3], Georgia[4], Kentucky[5], Texas[6], California[7], Missouri[8], and Massachusetts[9]. The total number of cases was relatively small in these reports and the largest number was in the southern states, probably due to immigrant carriers. To our knowledge there have been no previous cases of favus reported in Oklahoma, although our neighboring state of Texas reported two in 1949[6]. Shelmire stated that he had not seen a case of favus in Texas in 20 years. Pipkin[10] cited two of our cases in a recent paper.

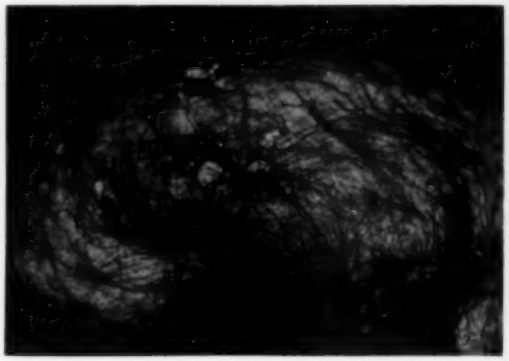
These above-mentioned facts, plus the additional fact that our cases (numbering six) have all been seen at the University Hospital in Oklahoma City during the past three years, indicate to us that these should be reported and that we should be cognizant of the clinical features in order to combat this recalcitrant type of tinea capitis. These cas-



RIGHT:

Illustration showing Tinea Capitis due to Trichophyton Schoenleini (Favus)

es are all from Oklahoma with no foreign extraction or contact with each other. Each family lives in an extreme section of the state from the other patients. The usual fungicides are ineffectual and x-ray epilation is almost mandatory for cure.



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Case	Sex and Age	Duration	Clinical Description	Examination Under Wood's Light	Treatment
No. 1 M.S. Choctaw County	F 8 yrs.	4 yrs.	Numerous manifestations of thick, adherent crusts over scalp. 3 to 4 cup-shaped, bright-yellow mounds of crust. Typical "mouse nest" odor to the scalp.	Dull-gray fluorescense of practically every hair of scalp.	Equal parts of ammoniated mercury and salicylic acid ointment, 10 per cent each. Poor cooperation at home.
No. 2 M.S. Choctaw County	F —6 yrs.	2 yrs.	Multiple, circinate, elevated, crusted, yellowish lesions of scalp with patchy alopecia.	Dull-gray fluorescence of all hairs with glowing of some of the crusts.	X-ray epilation of hairs of scalp using five-point Adems-Kienbock technique. Patient is clear to date.
No. 3 F.S. Choctaw County	M 33 yrs.	Lifetime	Crusting alopecia of posterior half of scalp with typical "mouse nest" odor. No Scutulae noted.	Dull-green fluorescence of each hair of posterior half of scalp.	Equal parts of ammoniated mercury ointment 10 per cent salicylic acid, 10 per cent salnidol ointment. Poor Cooperation.
No. 4 S.M. Jefferson County	F 9 yrs.	1 yr.	Almost complete alopecia with only scattered areas of tufts of hair present. Denuded atrophic areas over the scalp with typical scutulne present and "mouse nest" odor.	Practically every hair revealed a dull, bluish fluorescence.	Boric acid ointment 10 per cent locally; daily shampoos; ammoniated mercury ointment 10 per cent and salicylic acid ointment 10 per cent — equal parts. 20 per cent improvement. Poor cooperation.
No. 5 L.P. LeFlore County	F 18 yrs.	10 yrs.	Scalp contained multiple small, crusted lesions with definite loss of hair. Marked atrophy and cicatrization of the skin at the involved areas. Typical scutulne were present but without "mouse nest" odor.	Dull-gray fluorescence of all hairs of the scalp.	Daily shampoos and instructed how to epilate, using adhesive tape. Unable to get patient to return for any further treatment or observation.
No. 6 M.P. Muskogee County	F 4 yrs.	3½ yrs.	Large, thickened, lesions over scalp with bleeding on removal. Typical scutulae were present with definite "mouse nest" odor.	Du'l-green fluorescence of all hairs on scalp.	Furaspor (R) ointment locally; daily shampoos; instructions as to manual epilation of diseased hairs. When last seen (March 1952) patient exhibited 40 per cent to 50 per cent improvement.

LABORATORY

Microscopic examination of the individual hairs in 10 per cent KOH solution in each case revealed large spores in chains with attached hair "bubbles." Long hypheal filaments in the hairs had degenerated and although the outline of them remained, they were replaced by air. Cultures of the hair and scales on Sabouraud's media in each case revealed growth of the causative organism Achorion or *Trichophyton schoenleini.*

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VESTIBULAR DYSFUNCTION associated with

- streptomycin therapy

VERTIGO in

- Ménière's syndrome
- hypertensive disease
- fenestration procedures
- labyrinthitis
- radiation sickness

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

SEARLE *Research in the Service of Medicine*

President's Page

BUSINESS OR PROFESSION

No matter how you slice it, medicine is a business, but it is hedged in by some factors that set it apart.

In spite of some trite arguments to the contrary, ours is not a private business. For one thing, the tax payers advance many thousands of dollars for our education and if that is not an account receivable in their favor, then I don't know the meaning of decency. Because of our unique advantage, we also have a distinct moral obligation to society in the matter of fees and services. We can't in good conscience proclaim that our practice is a private matter to conduct as we please and charge all the traffic will bear, or to render service when it suits our convenience. In these respects we are definitely circumscribed.

Horse traders are presumably on equal footing, so it is each man for himself and the devil take the hindmost. But this can never be rightly true in medicine.

Patients themselves make it difficult for us to be professional men rather than merchants. "How much will your x-rays cost?" "Are your B.M.R.'s $5.00 or $10.00?", and, "Will there be a special rate on Tuesday?"

After a day in the office where she had received all sorts of tests and studies, a woman demanded an itemized bill, to which I replied that she was charged for only one item, namely, our professional opinion. If I contract for a chicken coop and it is delivered according to specifications, it is of no concern to me what tools the carpenter used in making it. To be sure a job requiring an adze would require more than one that can be done with a grubbing hoe.

We will never recapture the esteem our fathers enjoyed if we allow our vocation to be strictly a business and particularly if we insist that it is entirely a private business run solely for our own advantage.

Alfred R. Snuggs

President

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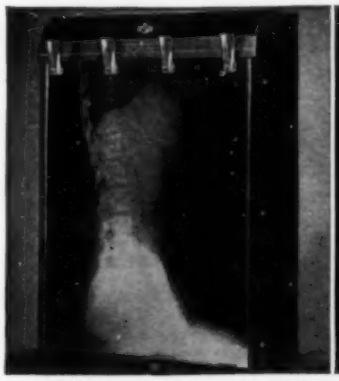

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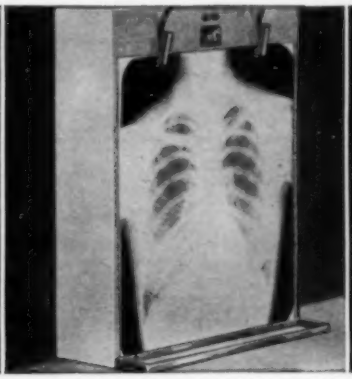

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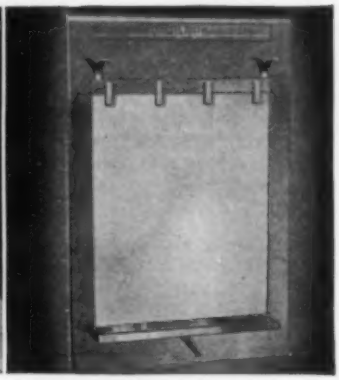

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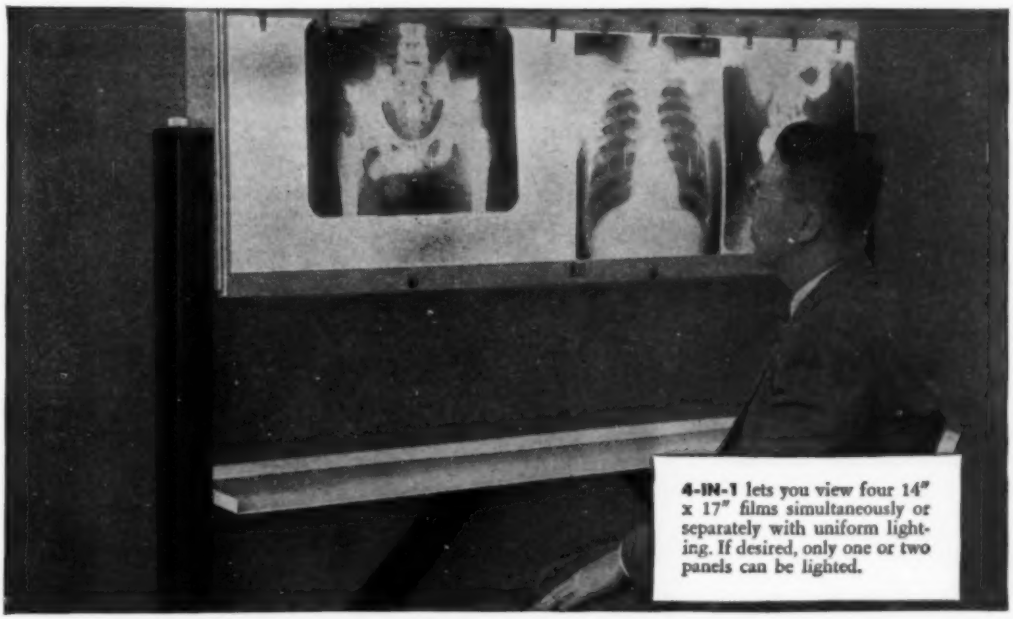

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OKLAHOMA ACADEMY OF GENERAL PRACTICE FIFTH ANNUAL MEETING TO FEATURE SCIENTIFIC PROGRAM, SEMINAR, ROUNDTABLE LUNCHEONS, DINNER-DANCE, TOUR

Oklahoma physicians are invited to attend the fifth annual meeting of the Oklahoma Academy of General Practice to be held at the Biltmore Hotel, Oklahoma City, February 16 and 17, 1953. The two-day meeting will be followed by a symposium on infectious diseases February 18, co-sponsored by the Academy and Lederle Laboratories.

At the time the February Journal went to press, program was still in the tentative stage and was as follows:

Monday, February 16

9:00 a.m. Stewart G. Wolf, M.D., Oklahoma City
"Custom Versus Facts in the Treatment of Peptic Ulcer"

9:30 a.m. Paul Williamson, M.D., Memphis, Tenn.
"Arteriosclerosis"

10:00 a.m. Frank R. Peterson, M.D., Cedar Rapids, Ia.
"Complications and Sequellae Following Cholecystectomy"

10:30 a.m. Break

10:45 a.m. J. Milton Singleton, M.D., Kansas City, Mo.
"The Earlier Diagnosis of Carcinoma of the Cervix"

11:15 a.m. Arild E. Hansen, M.D., Galveston, Texas
"Pertinent Points on the Rheumatic Fever Problem in Childhood"

11:45 a.m. Break

12:15 p.m. *Luncheon and roundtable*

2:00 p.m. *Seminar on family relations*

4:30 p.m. Tour of the Oklahoma Medical Research Foundation

6:30 p.m. Social hour.

7:30 p.m. Dinner and Dancing. Speaker at the dinner will be Mac Cahal, Executive Secretary of the American Academy of General Practice, whose topic will be *"The Public is Your Patient."*

Tuesday, February 17

7:00 a.m. *Breakfast*

9:00 a.m. Louis J. Levy, M.D., Fort Worth, Texas
"Fractured Ankles"

9:30 a.m. Arild E. Hansen, M.D., Galveston, Texas
"Office and Hospital Management of Infantile Diarrhea"

10:00 a.m. Paul Williamson, M.D., Memphis, Tenn.
"The Psychoneurosis, its Diagnosis and Treatment"

10:30 a.m. Break

10:45 a.m. Frank R. Peterson, M.D., Cedar Rapids, Ia.
"Certain Benign Lesions having Malignant Potentialities"

11:15 a.m. J. Milton Singleton, M.D., Kansas City, Mo.
"Diagnostic and Treatment Measures in Sterility"

11:45 a.m. Break

12:15 p.m. *Luncheon and Roundtable*

2:00 p.m. Hugh Payne, Executive Secretary, Oklahoma Medical Research Foundation
"Inside Glimpse into the Oklahoma Medical Research Foundation"

2:30 p.m. Stewart G. Wolf, M.D.
"The Mechanisms Underlying Epigastric Symptoms"

3:00 p.m. Louis J. Levy, M.D., Fort Worth, Texas
"Backache"

3:45 p.m. *Business Meeting*

SCHOOL SLATES OPEN HOUSE TO OBSERVE 60TH ANNIVERSARY

The University of Oklahoma School of Medicine has scheduled an open house for Sunday afternoon, February 8.

"Oklahomans will have an opportunity to see what is being accomplished at the School of Medicine," Dr. Mark R. Everett, dean of the school said. "Conducted tours will be held to enable the public to inspect the building and its facilities."

The open house is part of the week-long celebration held in connection with the University of Oklahoma's Sixtieth anniversary.

One o the highlights of the epen house will be the dedication of the new additions to the Scholo of Medicine building and university officials will be present for the ceremony. Alfred R. Sugg, M.D., Ada, will represent the Oklahoma State Medical Association.

The new additions have doubled the floor space of the School of Medicine building. These additions, which were completed in 1951, include the School of Medicine library, three large air-conditioned classrooms, student laboratories and department facilities.

"With the increased floor space for the scientific laboratories as well as the expansion of University hospitals," Dean Everett continued, "the school has been able to educate more students. In the school year of 1950-51 we had 280 medical students. We have 361 medical students this year and will be able to accommodate about 400 students in two more years.

"Of cvourse, the end result of these increased facilities will be more, beter-trained physicians for the communities of Oklahoma in the future," he concluded.

The original structure of the present School of Medicine building, located at Northeast Thirteenth and Phillips Streets in Oklahoma City, will round out a quarter of a century of service this year. Construction started in 1927 and was completed in 1928.

The School of Medicine was established at Norman in 1900. However, only the first two years of a full medical course were offered. In 1910, Epworth Medical College of Oklahoma City was merged with the OU School of Medicine. Under this arrangement, the first two years were taught at Norman and the last two years were taught at Oklahoma City.

Since 1928, the entire four-year curriculum has been offered on the Oklahoma City campus.

ANNOUNCEMENT TO PHYSICIANS OF OKLAHOMA

As Part of Its Program of Post-Graduate Medical Education,
The Oklahoma Academy of General Practice, in Conjunction with
Lederle Laboratories, Takes Pleasure in Inviting Oklahoma Physicians
To a Symposium on ACUTE INFECTIOUS DISEASES at

The Biltmore Hotel, Oklahoma City
Wednesday, February 18, 1953

Medical Sessions—10 am - 12:30 pm
2 pm - 5 pm

Papers will be presented on Mumps, Poliomyelitis,
Infections of the Urinary Tract, Infectious Mononucleosis,
Rabies, Pertussis, Measles, and Atypical Pneumonia

Luncheon and Dinner Will Be Served

Wives of Physicians are Cordially Invited to the Social Functions

There Will Be No Registration Fee

THE DALLAS SOUTHERN CLINICAL SOCIETY

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March 16, 17, 18, 19, 1953

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RICHARD H. FREYBERG, M.D., Internal Medicine, New York City

ROSS GOLDEN, M.D., Radiology, New York City

FRANK H. LAHEY, M.D., Surgery, Boston

NORMAN F. MILLER, M.D., Obstetrics-Gynecology, Ann Arbor

ALTON OCHSNER, M.D., Surgery, New Orleans

PAUL A. O'LEARY, M.D., Dermatology, Rochester, Minnesota

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HOTEL RESERVATIONS URGED FOR 60th ANNUAL MEETING IN APRIL

Program plans for the 60th Annual Meeting of the Oklahoma State Medical Association in Tulsa, April 13-15, 1953, were nearing completion last month as Dr. Gifford H. Henry, General Chairman, urged Oklahoma doctors to write at once for hotel accomodations.

Pointing to an outstanding scientific program highlighted by the appearance of 10 nationally known medical personalities as visiting distinguished guest speakers, Doctor Henry suggested that members planning to attend use the special reservation card included in this issue of *The Journal* to make reservations.

All requests for hotel accommodations will be handled through the Hotels Committee of the Tulsa County Medical Society in cooperation with the Tulsa Chamber of Commerce. Doctors may save time by writing directly to the Committee as requests sent to hotels will be referred to the Committee for disposition. Please state a first and second choice of hotels, dates of arrival and departure, and type of accommodations desired.

Dr. Rayburne W. Goen, Chairman of the Hotels Committee, said desirable accommodations are available at all major Tulsa Hotels. The meeting is to be at the Cimarron Ballroom. Hotel headquarters will be at The Mayo, where the House of Delegates will meet on Sunday, April 12, 1953. General sessions open on Monday, April 13th, and continue for three days. The Mayo and Adams hotels are located within one block of the Cimarron Ballroom. Other hotels are within walking distance.

Guest speakers for the 60th Annual Meeting will include:

Dr. W. Alton Ochsner, Professor and Chairman of the Department of Surgery, Tulane University School of Medicine, New Orleans, La.

Dr. F. Bayard Carter, Professor and Chairman of the Department of Obstetrics and Gynecology, Duke University School of Medicine, Durham, N.C.

Dr. Garfield G. Duncan, Professor of Medicine, Jefferson Medical School, Philadelphia, Pa.

Dr. Paul H. Holinger, Associate Professor of Otolaryngology, Northwestern University School of Medicine, Chicago, Ill.

Dr. Allan P. Bloxsom, formerly Professor and Chairman of the Department of Pediatrics, Baylor Medical College, Houston, Texas.

Dr. Carl A. Moyer, Professor of Surgery, Washington University School of Medicine, St. Louis, Mo.

Dr. James C. Sargent, Professor and Head of the Department of Urology, Marquette University School of Medicine, Milwaukee, Wis.

Dr. Francis J. Braceland, Professor of Psychiatry, Yale University School of Medicine, and Psychiatrist-In-Chief, The Institute of Living, Hartford, Conn.

Dr. Wendell G. Scott, Associate Professor of Radiology, Washington University School of Medicine, St. Louis, Missouri.

One other speaker will be announced later.

Complete ticket information concerning the President's Annual Dinner Dance will be announced in the March issue of *The Journal*. Featuring the nationally known music of Ralph Flanagan and His Orchestra, the Dinner Dance is expected to attract a capacity attendance. There will be an advance ticket sale.

MEDICAL SOCIETIES AROUND THE STATE

Atoka-Bryan-Coal

At the annual election of officers, LeRoy Engles, M.D., Durant, was elected president of the Atoka-Bryan-Coal Society. He succeeds Charles S. Moore, M.D. R. L. Cochran, M.D., Caddo, was elected vice-president and Seals L. Whitely, M.D., Durant, was named secretary-treasurer.

Oklahoma

Walter H. Dersch, M.D., Oklahoma City, is the 1953 president of the Oklahoma County Medical Society. New officers are Henry G. Bennett, M.D., president-elect; Lewis Carroll Taylor, M.D., vice-president; and Bert Mulvey, M.D., secretary-treasurer. Outgoing president is Ralph A. Smith, M.D.

East Central

Robert H. Furman, M.D., head of clinical research in cardiology at the Oklahoma Medical Research Foundation, was guest speaker at a recent meeting of the East Central Oklahoma Medical Society held at the Severs Hotel in Muskogee.

Creek

D. L. McAllister, M.D., Bristow, was elected 1953 president of the Creek County Medical Society. Other officers elected are Philip Joseph, M.D., Sapulpa, vice-president; and Charles Kent, M.D., secretary-treasurer. Guest speaker at the meeting was Allen Greer, M.D., Oklahoma City.

Grady

Succeeding Wesley W. Davis, M.D., Chickasha, Richard G. Stoll, M.D., Chickasha, was named 1953 president of the Grady County Medical Society. Other officers, all from Chickasha, are H. H. Macumber, M.D., vice-president; and Charles W. Ohl, secretary-treasurer.

Beckham

O. C. Standifer, M.D., Elk City, was elected president of the Beckham County Medical Society for 1953. M. J. Sugarman, M.D., Elk City, is vice-president and James W. Parker, M.D., also of Elk City, is secretary-treasurer. At the meeting for election of officers, the scientific program was presented by Richard Burgtorf, M.D., Shattuck, who spoke on "Fractures of the Upper Extremities."

Garfield-Kingfisher

Hope Ross, M.D., Enid, was elected 1953 president of the Garfield-Kingfisher County Society to succeed B. J. Cordonnier, M.D., also of Enid. John R. Taylor, M.D., Kingfisher, was elected vice-president and Roscoe C. Baker, M.D., Enid, was re-elected secretary-treasurer.

Cleveland-McClain

Charles A. Smith, M.D., superintendent of Central State Hospital, Norman, is the new president of the Cleveland-McClain County Society. Other officers are Kirk T. Mosley, M.D., vice-president; and Jim Haddock, secretary-treasurer.

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CURRENT ACTIVITIES AT THE OKLAHOMA MEDICAL RESEARCH FOUNDATION

EDWARD C. REIFENSTEIN, JR., M.D.
DIRECTOR

This is the fifteenth in the series of monthly articles concerning the current activities of the Oklahoma Medical Research Foundation. This article describes the investigative program in the field of cardiovascular disorders. The work of the Cardiovascular Section is supported by funds from the Oklahoma Medical Research Foundation, the Oklahoma State Heart Association, the Noble Heart Fund, and the National Heart Institute. The latter grant in the amount of $14,000.00 (December 1952-November 1953) was awarded recently.

The Cardiovascular Section was activated when Dr. Robert H. Furman, formerly Assistant Professor of Medicine, Nashville, Tennessee, assumed his duties as head of the Section on April 1, 1952 (see *J. Okla. State Med. Assoc. 45:30,* Jan. 1952). In July, 1952, Dr. Loyal Lee Conrad, formerly Chief of the Oklahoma University Hospital Clinical Pathology Laboratories, became associated with Doctor Furman in the Cardiovascular Section (*see J. Okla. State Med. Assoc. 45:430,* Dec. 1952).

While much of the effort to date has been concerned with acquiring ancillary personnel and developing techniques and instrumentation, actual research work has been under way since July 1952. Most of this has been in the field of atherosclerosis.

Search has been made for a simple method for the quantitation of "clearing factors" or "antilipemia substances" which appear related to, if not necessary for, the stable and orderly transport of serum lipids. It is postulated that aberrations in the clearing or antilipemic properties of plasma may be related to atherogenesis. Preliminary studies indicate that various stable emulsions prepared by the use of high pressure viscolizers may be utilized to demonstrate the clearing property of plasma. Clearing is measured by changes in the optical density of the emulsion. The pH of plasma is being measured to determine whether or not clearing is associated with the release of fatty acids, and plasma has been tested from fasting subjects before and following the injection of heparin. In addition, studies utilizing paper and/or zone electrophoresis techniques are planned in an effort to determine whether or not changes in the electrophoretic pattern of plasma proteins can be related to changes in the clearing properties. A significant deviation from the "normal" has been noted in a group of diabetic subjects.

Preparative, analytical and ultracentrifuges are scheduled for arrival at the Oklahoma Medical Research Foundation in December, 1952, or January, 1953, and, when these instruments have been calibrated and sufficient technical experience has been gained by the operators, an attempt will be made to determine whether or not there exists any correlation between antilipemia factor or factors and the lipoprotein pattern of the plasma. The practical application of these studies will be to provide a partial answer to the following questions:

1) Can plasma emulsifying and stabilizing agents be simply quantitated?

2) Is there any correlation between such emulsifying and stabilizing agents and the presence or absence of abnormal lipoproteins?

3) Is there any correlation between the presence or absence of these agents and atherosclerosis?

4) What are these stabilizing agents?

5) Can they be separated from plasma?

6) Can they be made available for therapeutic use?

In addition to these studies, various therapeutic regimens will be evaluated, including the use of steroid compounds, lipotropic substances, metabolically active hormones and dietary regulation on the lipoprotein pattern and antilipemic activity of plasma from human subjects. The ultracentrifuge, paper electrophoresis and optical density measurements will be utilized in this investigation.

Studies in experimental atherogenesis in dogs have been under way for several months, with some very interesting results. A toxic amine substance, known to cause necrotizing arterial lesions when given in sufficient quantity, when administered in small amounts produces two types of lesions, one a proliferation of the intimal cells, and the second a ground-substance lesion resembling that seen by certain observers early in human atherosclerosis. This experimental lesion will be used to assay the value of various substances as prophylactic agents against the development of atherosclerosis. Clinical application of such agents is the practical goal of these studies.

A second major field of endeavor concerns cardiovascular and pulmonary dynamics. Initial studies in patients with pulmonary emphysema have indicated that in selected cases the use of artificial pneumoperitoneum has a physiological basis and a therapeutic value. Since the basic pulmonary lesion in pulmonary emphysema is over-distention of the lngs, a project is planned in which partial, controlled pneumothorax will be used in addition to pneumoperitoneum in order to reduce the volume of the lungs. Such cardiopulmonary dynamic studies will utilize cardiac catherterization, lung volume and ventilatory function measurements. The tooling-up for this investigation has been long and expensive, but the initial phases of this study have been completed, and definitive work will be started on delivery of direct-writing, recording galvanometers, expected shortly. The problem of pulmonary emphysema is unusually important because to date therapeutic measures have been largely ineffective, and because ever increasing numbers of individuals are entering the "old age group" and developing this disorder.

A relatively new application of the cardiac catheter is planned. This involves the use of radio-opaque plastic catheters, placed intra-arterially, through which the body organs in situ can be perfused. Collaborative studies are planned with the members of the Endocrinology and Metabolism, Biochemical and Cancer Sections of the Research Institute. By this intra-arterial technique certain therapeutic agents such as nitrogen mustard, triethylenemelamine, and aminopterin may be injected directly into organs with various neoplastic diseases.

An experimental therapeutic approach to pulmonary embolism has been devised and is now being considered

(Continued on Page 52)

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HAVE YOU HEARD?

Alfred R. Sugg, M.D., Oklahoma State Medical Association President from Ada, was named to the Board of Directors of the Oklahoma Public Expenditures Council December 11.

D. D. Cramblett, M.D., formerly of Stigler, has recently moved to Holdenville.

Ray J. Camp, M.D., formerly of Woodward, is taking a three year surgical residency in Minneapolis.

J. T. Colwick, M.D., Durant, recently took a postgraduate course in general surgery in Dallas.

Frank E. Flack, M.D., Woodward, has opened his offiices at 1218 Eighth St., Woodward.

Roger Reid, M.D., Ardmore, has been elected president of the staff of Hardy Sanitarium there.

L. B. Word, M.D., Bartlesville, was guest speaker at a DeMolay meeting in that city recently. His topic was "Sex."

M. Wilson Mahone, M.D., Hobart, has been named part-time medical consultant for the Kiowa county health department.

J. M. Newton, M.D., formerly of Massachusetts, has been named medical director for the Oklahoma State hospital at Clinton.

John B. Jacob, M.D., formerly of Waurika, has been recalled to active duty with the navy. He reported to Memphis, Tenn.

Henry Laurens, Jr., M.D., Ada, has been notified of his election to the American Gastroscopic Society.

M. H. Newman, M.D., Shattuck, has been appointed to the Board of Medical Examiners to succeed his father, O. C. Newman, M.D., who resigned, for the unexpired term.

R. M. Howard, M.D., Oklahoma City, has been selected as the Oklahoma physician to be sent to the World Medical Association first western hemisphere conference in Richmond, Va. in April by the A. H. Robins Co. with all expenses paid.

CURRENT ACTIVITIES

(Continued on Page 50)

for financial support by a national organization. Radiographic and fluoroscopic equipment is needed to implement this study, but once this equipment is available the project can be initiated promptly and should reveal definitive results in a very short period of time. If the proposed technique proves feasible it may alter markedly the current method of managing pulmonary embolism. This interesting project will be discussed more fully at another time.

In addition to the investigations outlined above, certain ancillary studies with other Sections of the Foundation are planned. The members of the Cardiovascular Section are responsible also for the cardiac catheterization of patients on the medical service of the University Hospital. In addition to these duties, the professional members of this Section have teaching responsibilities at both the medical student and postgraduate levels. These duties directly related to the program of the Medical School and the University Hospital involve over 20 per cent of the average working time of the members of the Cardiovascular Section.

HISTORY OF MEDICINE COMMITTEE TO MEET

Plans are being made for a meeting of those interested in the history of medicine in Oklahoma. The original meeting of the group was scheduled for January 25 but was postponed because of inclement weather. Physicians from each of the counties were invited to be present along with representatives of the Department of Archives at the University of Oklahoma, the President of the University, Dean of the Medical School, and others. This meeting was to initiate the activities that had been approved by the House of Delegates at its last meeting. Each county society and individual member is urged to cooperate and those interested are asked to contact the Executive Office before the next meeting of the group which will be held sometime following the celebration of the 60th Anniversary activities at the University of Oklahoma, Norman.

Physicians with interesting historical documents, etc. are especially asked to notify the Executive Office or Lewis J. Moorman, M.D., chairman of the History of Medicine Committee of the Association.

DIABETES GROUP TO MEET

Oklahoma physicians are urged to attend a meeting of the Oklahoma State Diabetes Association February 15, 1953 at the Biltmore Hotel at 6:00 p.m. A charge of $5.00 per person will be made and will include the social hour, dinner and address by Stewart Wolf, M.D., Professor of Medicine at the University of Oklahoma School of Medicine. Doctor Wolf's topic will be "Life Stress and the Diabetic Metabolism."

Reservations may be made through Dr. R. Palmer Howard at the Oklahoma Medical Research Foundation, 825 N.E. 13th St., Oklahoma City, Okla. Reservations, accompanied by a check in the amount of $5.00, should be made by February 14. Hugh Jeter, M.D., Oklahoma City, is president of the group.

CLASSIFIED ADS

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